# Knock-On Effects of Hypothyroidism Lead to Permanent Alternation to Adenine Transport Molecule Manufacturing Process Leading to Treatment Resistance

17 October 2025
Simon Edwards
Research Acceleration Initiative

## Introduction

Many patients fail to fully respond to conventional treatments for hypothyroidism, sc. T4 supplementation. Patients may live for many years with hypothyroidism before a proper diagnosis may be made and therein may lie the explanation for why so many fail to fully respond to T4 supplementation.

## Abstract

Tri-iodothyronine is critical for supporting cellular metabolism. Whereas insulin is associated with sugar metabolism, tri-iodothyronoine could arguably be said to be more closely associated with oxygen metabolism. Sugar and oxygen must ultimately combine to support ATP production and at the heart of this is the need to manufacture and transport a molecule called adenine.

I propose that the key to understanding the etiology of certain idiopathic cases treatment-resistant hypothyroidism lies in studying the reaction of those patients to caffeine. When a healthy person consumes caffeine, which is structurally quite similar to adenine, the result is the stimulation of the metabolism. Caffeine, however, is not adenine and cannot actually be used by the body. Nonetheless, it has a stimulant effect because caffeine, acting as a pseudo-adenine, disrupts the body's own ability to utilize adenine. This forces the body to overproduce adenine to crowd out the caffeine and to leverage more of its free T3. For someone with healthy thyroid function, caffeine causes an increase in available metabolic energy followed by a crash, usually while the person is sleeping when it is not noticed.

However, something interesting might be predicted to happen when an individual with hypothyroidism which is treatment-resistant consumes caffeine. Because the body's system for transporting adenine may be impaired, the adenine transport system is already functioning maximally and does not enjoy a benefit from the introduction of a pseudo-adenine. If anything, the pseudo-adenine's presence, in the case of those patients, reduces the overall ability to utilize adenine because the pseudo-adenine crowds out the adenine and the body has no auxiliary capacity, in the case of those patients, to perform a compensatory action.

I posit that the unavailability of T3 over time results in permanent epigenetic changes which cause the body to employ a truncated process for manufacturing a molecule which is responsible for the transport of adenine. This incomplete molecule actually does a poorer job of latching onto adenine, to such an extent that a person's metabolism would be better served by making fewer of the complete molecules than more of the incomplete

molecules.  However, the body does not know this and the switches are thrown in response to the chronically low T3 levels resulting in this truncated process.

After a patient begins taking thyroid supplements, part of the overall problem is addressed (T3 and T4 levels are restored to the proper level and TSH is reduced, preventing undue thyroid cancer risk and available adenine is restored,) however, the overall rate of ATP production at the cellular level may remain impaired due to the malformed transport molecule (molecule unknown but hypothesized to exist by this author.)

**Conclusion**

If this hypothesis holds true, it should be possible to use an mRNA approach to throw the switches back so that the body reverts to the proper process of manufacturing this adenine transport molecule so that available ATP may be restored to the proper level.  In the interim, patients experiencing this unnamed condition, which is not consistent with either Reverse T3 Syndrome or Hashimoto’s Thyroiditis, might be advised to avoid caffeine as this clearly only further frustrates an already-impaired adenine transport process.